undecided.[1] I found similar responses amongst a random sample of 1000 general practitioners in New Zealand: 22% reported that informed consent for testing was not at all important and 4% were undecided. I, too, found that years since graduation was a significant variable as was the number of patients requesting an HIV test. Younger doctors and those with the most requests for tests believed more strongly in informed consent.

In addition I examined attitudes to anonymous testing and to confidentiality. Anonymous testing, although recommended and widely practised in New Zealand, was disagreed with by 28% of the sample, including 7% who disagreed strongly. In fact, only 41% agreed with it, the remainder being neutral. Attitudes to confidentiality were shown by the doctors' responses to questions on sharing information about patients with AIDS. Sixteen per cent of the doctors would give such information to reception staff, and the same proportion would give it to colleagues outside the practice. Women doctors were less likely to do this than men.

It is worrying that the findings of this study and those of Dr Shapiro show some indifference to major issues regarding patients' rights such as informed consent and anonymous testing. This is particularly sad given the crucial role that general practitioners will have in the future in caring for those with AIDS or HIV infection and their families. Such indifference will inevitably lead to mistrust on the part of patients and to a reluctance to seek help from general practitioners when it is needed. Future efforts in educating general practitioners about HIV must address these matters.

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Passive smoking and middle ear effusion in children

SIR,—Dr D Strachan and colleagues presented an interesting report on the possible association of passive smoking with otitis media with effusion,[1] but there are three points that need addressing before their conclusions can be reached.

Eustachian tube dysfunction is extremely common in children and gives rise to negative middle ear pressure and middle ear effusion. The prevalence of middle ear effusion varies inversely with age, but there is also a marked seasonal variation, possibly related to a similar variation in upper respiratory tract infections.[2] The authors have quite rightly confined themselves to a single age group, but they carried out their tests over a period from January to June. Those tested earlier should have a higher rate of abnormal results, but this is not taken into account in the analysis.

Perhaps more importantly they fail to indicate whether the children had already had ear, nose, and throat operations (which at the age of 7 must be a considerable percentage) or whether there were other important factors such as cleft palate or Down's syndrome.

As otolaryngologists we deplore the use of tympanometry alone in the diagnosis of middle ear disease. It is a useful screening test, but in the presence of an abnormal finding we believe otoscopy must be performed. Frequently an obvious cause for the flat tympanogram such as wax, perforation, or even a grommet will be found. Dr Strachan and colleagues unfortunately do not seem to have checked their findings with otoscopy.

A possible indicator of this fault can be found in their numbers of abnormal tympanograms with increasing cotinine concentrations. There seems to be a trend relating increasing incidence of flat tympanograms to cotinine concentration, but there is no such trend with negative middle ear pressure. If one assumes that the same pathological process causes both negative middle ear pressure and middle ear effusion through dysfunction of the eustachian tube then the association between passive smoking and middle ear effusion is quite likely to be spurious.

It seems a pity that a paper written by an epidemiologist, a psychiatrist, and a chemist about an ear, nose, and throat condition should not have had the very necessary skills of an otolaryngologist to validate its findings.

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1 Strachan DP, Jarvis MJ, Feyerabend C. Passive smoking, salivary cotinine concentrations, and middle ear effusion in 7 year old children. *Br Med J* 1989;298:1549-52. (10 June.)
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AUTHORS' REPLY,—Impedance tympanometry may not be the definitive diagnostic test for middle ear diffusion, but most previous epidemiological surveys have relied on this technique. Tympanometric measurement of the physical volume of the ear canal guards against the common sources of error. High values (>2·0 ml) indicate a perforated eardrum or patent ventilation tube, and in the seven children with such abnormality we conservatively chose to analyse the tympanogram from the other ear. No results were recorded from ears with blockage of the probe or low physical volume (<0·5 ml) suggesting wax. It is unlikely that flat tympanograms attributable to impacted wax could have generated a spurious association with passive exposure to smoke.

Children whose tonsils or adenoids had been removed (n=104) were at substantially higher risk of middle ear effusion (22% *v* 8%). Such a history was unrelated to the presence of smokers in the household (12% *v* 13%), so it is unlikely that previous surgical treatment affected the observed relation between middle ear effusion and passive exposure to smoke.

Different relations of passive smoking to type C and type B tympanograms might be expected if tobacco smoke affects the persistence of effusions rather than their incidence. In fact, normal (type A) tympanograms were less common in the children with higher cotinine concentrations, so that among the children without effusion there was a slightly higher risk of reduced middle ear pressure with heavy exposure to smoke (table II in our paper).

Month of examination would not affect the association between tympanometric findings and the number of smokers in the household, which was ascertained by a simultaneous questionnaire survey, but it was a potential confounding variable in our analysis of middle ear effusion and salivary cotinine concentrations. The prevalence of type B tympanograms was higher among children tested in January or February (12%) than in March or April (10%) and May or June (7%). After adjustment for sex, housing tenure, and number of smokers in the household the geometric mean salivary cotinine concentration in January and February was approximately double that in May and June. Nevertheless, the relation between middle ear effusion and the logarithm of the cotinine concentration remained significant after adjustment for month of examination (odds ratio per doubling 1·12, 95% confidence interval 1·01 to 1·25, $\chi^2=4{\cdot}30$, df = 1). Indeed, after adjustment for log cotinine the trend in prevalence by month of examination was non-significant (χ^2 (trend)= 1·07, df = 1). Greater indoor exposure to tobacco smoke during the winter may contribute to the seasonal variation in prevalence of middle ear effusion.

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SIR,—I agree with Dr D P Strachan and colleagues that middle ear effusions in children should be recognised as one of the hazards of passive smoking.[1] It has been recognised for some time that passive smoking may have deleterious effects on the respiratory tracts of children. It is only recently that the adverse effects of passive smoking on the middle ear have been reported.

Not only is passive smoking in children associated with a higher than expected incidence of middle ear effusions and abnormal results of tympanometry[2] but these children are put at an increased risk of undergoing surgery for the condition. There is an increased incidence of grommet insertion and adenoidectomy in children whose parents smoke. Such children are twice as likely to require adenoidectomy, and their chance of requiring grommet insertion is increased by half.[3]

It is important to impress upon smoking parents that they may be subjecting their children not only to a greater risk of middle ear effusions but also to an increased likelihood of surgical intervention for the condition with the possibilities of both physical and psychological complications.

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Transurethral prostatic resection: a safe operation

SIR,—I was intrigued by Mr G Williams and colleagues' use of an expandable metal mesh stent for treating prostatic obstruction in patients considered unfit for transurethral prostatic surgery.[1] I was surprised, however, by their statement that the mortality associated with transurethral resection of the prostate has "led to a search for less invasive treatments," thus prompting this innovation.

Equally surprisingly, Mr Williams and colleagues were presented (in a short time, it would seem) with nine patients who were considered unfit for prostatic resection and hence were offered a stent as alternative treatment. Although the authors did not indicate the timespan over which these cases were collected, it could not have been very great as the first urological use of these stents was reported only in 1988. Accordingly the nine patients considered unfit for transurethral resection would seem to represent an uncharacteristically high proportion of all patients referred for prostatic surgery during this relatively short period. All this is at odds with my experience and that of my colleagues. I work in a 590 bed teaching hospital. In the 12 months to March 1989, 328 transurethral resections of the prostate were performed by the urology unit. None of the patients died, and only one patient with severe ischaemic heart disease was advised that he was unfit for surgery.

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